or eustachian tube dysfunction and may differ briefly from a child with SOM after acute otitis. We need more study of this group with chronic SOM.

In summary, based on these studies there is no supporting evidence thus far for using antihistamines, decongestants or their combinations for (1) the prevention of AOM when a cold develops, (2) the prevention of SOM when AOM is being treated or (3) the treatment of SOM once it develops.

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Yersinia Enteritis in Children

YERSINIA-ASSOCIATED gastroenteritis is caused by *Yersinia enterocolitica* or *Yersinia pseudotuberculosis*. Both organisms are related to the causative agent of plague, *Yersinia pestis*. The organisms may be confused with Gram-negative nonlactose-fermenting enteric pathogens but are distinguished by their growth in specifically defined media or under cold enrichment. *Y pseudotuberculosis* produces an invasive lesion in experimental animals with granulomas in mesenteric nodes. A heat-stable toxin that is biochemically similar to other enteric pathogens has been isolated from human strains of *Y enterocolitica* and the organism can also produce lesions of the lymphatic system of the gastrointestinal tract and mesenteric lymph nodes.

In the United States *Y enterocolitica* enteritis in children seems to be an infrequent cause of diarrhea. In a survey of enteropathogens at Children's Hospital National Medical Center in Washington from 1974 to 1976, only five cases of diarrhea were described with *Y enterocolitica*. Four of these five children had primary enteric illness, while the fifth patient had associated ocular and joint involvement. In three of five cases the disease was self-limited. Two of the five patients seemed to respond to antimicrobial therapy with either orally administered ampicillin or trimethoprim-sulfamethoxazole. In other series, the acute enteritis is characterized by fever (87 percent), diarrhea (69 percent), severe abdominal pain (62 percent), vomiting (56 percent) and other constitutional symptoms such as pharyngitis, headache and leukocytosis. Blood may be present in stools. The average illness lasts from one to two days and subsides spontaneously without sequelae. Yersinia-associated enteritis is reported most frequently in cold months. Both *Y pseudotuberculosis* and *Y enterocolitica* have been identified from a wide range of animal and environmental sources, including domesticated animals such as pigs, goats and dogs and contaminated animal products such as goat's milk, chocolate milk or contaminated water sources. Person-to-person spread appears to be unusual. The incubation period is between five and seven days for both of these pathogens.

Two other presentations of *Y enterocolitica* infection occur. One is a syndrome of abdominal pain more common in older children and adolescents. This illness generally begins with abdominal pain, fever and vomiting; diarrhea may not be a prominent feature. The pain often localizes to the lower quadrants and may mimic mesenteric adenitis, terminal ileitis or a true appendicitis. Intestinal perforation or metastatic spread of infection may develop. The other, less frequent, presentation of *Yersinia* infection is characterized by the development of systemic manifestations in addition to enteritis. The clinical spectrum may range from erythema nodosum with mild or moderate arthritis to involvement of the eye with iritis and conjunctivitis. Classical Reiter's syndrome may develop and appears to be associated with persons who possess the HLA-B27 antigen.

Yersinia-associated gastroenteritis should be suspected in any case of febrile diarrheal illness and in cases of acute abdominal pain simulating appendicitis. If mesenteric adenitis, appendicitis or ileitis is found at surgery, appropriate cultures should be taken. *Yersinia* can be isolated with cold enrichment (4°C for three weeks) and grows on MacConkey medium or Salmonella-Shigella medium incubated at 22° to 29°C. Antibody titers can be measured by agglutination or hemagglutination and may be available through public health laboratories. Maximum antibody titers are usually present at two weeks. Most strains of both pathogens are sensitive in vitro to chloramphenicol, the aminoglycosides, trimethoprim-sulfamethoxazole and tetracycline; susceptibility to the cephalosporins, semisynthetic penicillins and ampicillins is

variable. Although efficacy of antibiotics in treating Yersinia-associated enteritis has not been established, most would recommend therapy in severe infections.

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Toxic Shock Syndrome in Children and Adolescents

FIRST DESCRIBED by Todd and co-workers in 1978, the toxic shock syndrome is a new disease entity with well-defined epidemiologic and clinical criteria for diagnosis. In the original description, seven children presented with staphylococcal infection. Two of these children had focal staphylococcal abscesses, and local infection resulted in a fulminant disease that affected many organ systems. The disease today is recognized primarily in young, menstruating women using tampons. The incidence of this condition in other patients not associated with tampon usage is not known.

The clinical features and case definition criteria for toxic shock syndrome are as follows: (1) fever (temperature 38.9°C [102°F]), (2) rash (diffuse macular erythroderma), (3) desquamation (one to two weeks after onset of illness, particularly of palms and soles), (4) hypotension (less than fifth percentile by age for children 16 years of age or younger, or orthostatic syncope), (5) three or more of the following types of involvement: *gastrointestinal* (vomiting or diarrhea, or both, at onset of illness), *muscular* (severe myalgia or creatine phosphokinase twice normal), *mucous membrane* involvement (vaginal oropharyngeal or conjunctival hyperemia), *renal* (blood urea nitrogen or creatinine levels twice normal or urinalysis showing greater than five leukocytes per high-power field in the absence of a urinary tract infection), *hepatic* (total bilirubin, serum aspartate aminotransferase [formerly, serum glutamic oxaloacetic transaminase, SGOT] or serum alanine aminotransferase [formerly, serum glutamic pyruvic transaminase, SGPT] twice normal), *hematologic* (platelets less than 100,000 per cu mm), *central nervous system* (disorientation or alterations in consciousness without focal neurologic signs), negative results from *blood, throat* or *cerebral spinal fluid cultures; negative serologic tests* for Rocky Mountain spotted fever, leptospirosis or measles.

Most patients recover completely within 7 to 14 days. Desquamation may be patchy but occasionally involves almost the entire body. Hair loss and nonreversible nail defects have also been reported.

The incidence of toxic shock syndrome in menstruating women is estimated at 3 per 100,000 per year. Since this is a low figure compared with tampon usage, additional factors must play a role. The association with colonization or infection with phage group 1 *Staphylococcus aureus* is clear. If toxigenic *S aureus* is the causative factor, how does tampon use predispose to the disease? Investigations have shown that regular use of tampons and of so-called superabsorbent tampons produce vaginal mucosal drying and epithelial changes. The superabsorbent tampons may produce microulcerations that facilitate local extension of vaginal staphylococcal colonization to the circulatory system.

Toxic shock syndrome is a multisystem disease varying in severity and with a variety of historical, physical and laboratory manifestations. It is similar to other desquamative conditions such as Kawasaki's disease (mucocutaneous lymph node syndrome), the scalded-skin syndrome, cat scratch disease, scarlet fever, Rocky Mountain spotted fever, leptospirosis and staphylococcal food poisoning. The above conditions must be ruled out and can be excluded by appropriate laboratory tests. Retrospective analysis of other patients in the literature thought to have one of the above mentioned desquamative conditions now fulfill the criteria of toxic shock syndrome and the incidence of the disease will increase as more data are reviewed.

Investigations are now concerned with the production of a staphylococcal toxin that is probably responsible for the disease. The pyogenic and exfoliative toxins associated with *S aureus* infection have not been isolated consistently in patients with the toxic shock syndrome. Perhaps a new yet unidentified toxin is responsible for the clinical manifestations of the disease.

Therapy of toxic shock syndrome depends on the severity of the illness. Severe cases require intensive care facilities, with initial therapy directed toward correction of hypotension. Oncotic pressure should be monitored and vasopressors